Seasonal Differences in the Development of Heightened Resistance of the Isolated Heart during Adaptation to Physical Exercise

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Substantial seasonal differences are found in the development of the cardioprotective effect of adaptation to physical exercise: in winter such adaptation results in an increase of the resistance of the isolated heart to the contracture and arrhythmogenic effects of ischemia and reperfusion, while in summer the anticontracture effect is absent and the antiarrythmic effect is significantly lowered.

Key Words: *adaptation to physical exercise; heart; ischemia; reperfusion; seasonal differences*

The adaptation to intermittent physical exercise is known to have a marked effect on the resistance of the cardiovascular system to damaging factors. Such adaptation considerably enhances the resistance of the myocardium to ischemia and reperfusion [7,10], limits the region of ischemic necrosis [9], and prevents loss of rhythm and damage to the contractile function of the heart in experimental aortal stenosis [2] and severe emotional-pain stress [3]. The protective effect of adaptation was recently found to manifest itself not only under conditions of the whole organism but also at the level of the isolated heart [1]. At the same time, the seasonal differences in the development of direct cardioprotective effects of such adaptation remain largely unexplored, even though they are not only of theoretical significance but also important for optimizing the clinical use of adaptation to physical exercise in order to treat and prevent cardiovascular disorders.

The present investigation was undertaken to compare the cardioprotective effects of adaptation to physical exercise in winter with those adaptation in summer.

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MATERIALS AND METHODS

Experiments were carried out on male Wistar rats weighing 250-300 g. The adaptation to physical exercise (swimming) was spread out over 45 days as follows: the duration of swimming was increased from 15 to 30 min per day during the 1st week and then to 60 min per day during the 2nd week, after which it remained unchanged (60 min per day) till the end of the experiment. The water temperature was 32°C.

For assessment of the seasonal differences of the effects of adaptation one group of animals was adapted during the winter period (December - February), while the other was adapted during summer (June - July).

The cardioprotective effect was examined using the isolated heart technique. The heart was perfused after Langendorff with standard Krebs-Henseleit solution. The mechanical activity of the isolated heart was determined with a TD-112S isotonic transducer (Nihon Kohden). The mechanical activity and electrocardiogram were recorded with RM-6000 polygraph modules (Nihon Kohden). Ischemic and reperfusion damage was simulated by totally cutting off the coronary flow for 20 min, after which perfusion was resumed and observations were continued

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for 15 min. The protective effect was assessed in percent by the degree of inhibition of depression of the amplitude of contraction and by the degree of prevention of contracture and arrhythmia of the isolated heart in ischemia and reperfusion. Total cessation of the damaging effect of ischemia or reperfusion was taken as 100%.

The results were processed statistically using the Student *t* test.

RESULTS

Ischemia followed by reperfusion resulted in a marked contracture, depression of the contraction amplitude, and rhythm disorders in control animals both in summer and in winter (Table 1). In the group of animals adapted in winter the ischemia-induced contracture was practically absent and contraction in response to reperfusion was lowered by 60% as compared to the control. The amplitude of contraction in the hearts of these adapted animals was 6 times higher than in control. The anticontracture effect of winter adaptation was accompanied by antiarrhythmic activity. For example, the overall duration of tachycardia and ventricular fibrillation calculated per heart was 6.8±2.2 min in the control versus 1.8±1.7 min for adaptation to physical exercise. Ventricular fibrillation developed in 5 out of 8 hearts in the control but only in 1 out of 8 hearts in the case of adaptation. Therefore, winter adaptation to physical exercise has marked anticontracture and antiarrhythmic effects and clearly protects the isolated heart from both ischemia and reperfusion damage.

Summer adaptation has a different pattern of effects. The marked anticontracture action found in winter was completely absent in summer. Restriction of the depression of the contraction amplitude in reperfusion was insignificant and unreliable in summer-adapted animals (Fig. 1). Summer adaptation also limited the development of ventricular fibrillation and tachycardia, but the antiarrhythmic effect was much less pronounced than in winter (Table 1, Fig. 1).

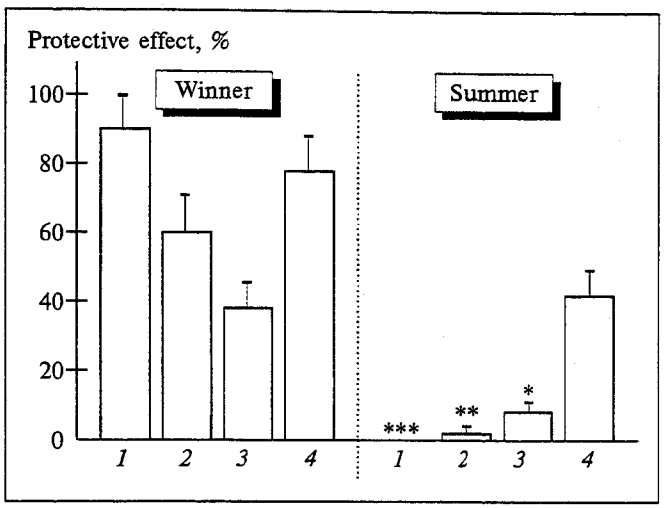

Fig. 1. Seasonal differences in the development of the cardioprotective effect of adaptation to physical exercise. Complete prevention of damage from ischemia and reperfusion is taken as 100%. *1*) restriction of isolated heart contracture in the 20th min of ischemia; *2*) restriction of isolated heart contracture in the 5th min of reperfusion; *3*) restriction of depression of contraction amplitude in the 5th min of reperfusion; *4*) decrease in the number of hearts developing ventricular fibrillation in response to reperfusion. Significance of differences between the cardioprotective effect of adaptation in summer and winter: $^{*}p<0.05$, $^{**}p<0.01$, $^{***}p<0.001$.

Thus, the findings attest to seasonal differences in the development of a direct cardioprotective effect of adaptation to physical exercise: in winter such adaptation increases the resistance of the isolated heart to contracture and arrhythmia induced by ischemia and reperfusion, whereas in summer the anticontracture effect is absent and the antiarrhythmic effect is significantly lowered.

The data may be useful in the clinical setting, where the seasonal differences should be taken into account when using physical exercise to enhance resistance to ischemia and reperfusion.

TABLE 1. Effect of Summer and Winter Adaptation to Physical Exercise on Resistance of Isolated Heart to Ischemia and Reperfusion

Parameter	Winter period		Summer period	
	control (n=8)	adaptation (n=8)	control (n=7)	adaptation (n=6)
Initial contraction amplitude, mm	2.7±0.1	2.7±0.2	3.2±0.1	3.1±0.2
Contracture in 20th min of ischemia, mm	1.3±0.3	0.02±0.02**	1.8±0.3	1.8±0.2
Contracture in 5th min of reperfusion, mm	1.8±0.2	0.7±0.1*	2.3±0.2	2.1±0.3
Contraction amplitude in 5th min of reperfusion, mm	0.2±0.1	1.2±0.1**	0.3±0.1	0.5±0.1
Percentage of hearts developing ventricular fibrillation in reperfusion	62.5	12.5*	57.1	33.3

Note. Significance of differences between control and adaptation: $^{*}p<0.05$, $^{**}p<0.01$.

There is no explanation so far for the seasonal differences in the effects of adaptation to physical exercise. We may speculate that they have to do with the seasonal activation of the central and local mechanisms of adaptation, such as boosting of the energy-supplying systems of the myocardium [11,13], of the antioxidant systems [12], and of the Ca^{2+}-transporting systems [8]. In fact, myocardial contractility and arterial pressure were found to be higher in rabbits in the spring-summer period than in winter, while the plasma content of fatty acids was significantly lower in summer [5]. In addition, ultrastructural study of the myocardium revealed an elevated number of mitochondria and cristae in summer, but a greater volume of mitochondria in wintertime [6]. Significant seasonal fluctuations in antioxidant system activity are found in human erythrocytes [4].

REFERENCES

1. I. Yu. Malyshev, P. A. Prodius, and F. Z. Meerson, *Byull. Eksp. Biol. Med.*, **119**, №1, 25-28 (1995).
2. G. I. Markovskaya, T. F. Shevchenko, O. N. Lyubimtseva, and S. I. Shaginova, *Kardiologiya*, № 5, 77-80 (1973).
3. F. Z. Meerson and V. V. Malyshev, *Ibid.*, № 2, 74-77 (1985).
4. S. N. Suplotov and E. N. Barkova, *Lab. Delo*, № 8, 459-463 (1986).
5. V. A. Frolov, L. V. Efimov, T. A. Kazanskaya, and V. N. Kozyrenko, *Dokl. Akad. Nauk SSSR.*, **249**, № 2, 504-508 (1979).
6. V. A. Frolov, *Byull. Eksp. Biol. Med.*, **97**, № 4, 420-424 (1984).
7. M. M. Bersohn and J. Scheuer, *Amer. J. Physiol.*, **234**, H215-H218 (1978).
8. H. Guski, F. Z. Meerson, and G. Wassilew, *Exp. Pathol.*, **20**, 108-120 (1981).
9. J. E. Koerner and R. L. Terjung, *J. Appl. Physiol.*, **52**, 376-387 (1982).
10. T. D. Noakes, L. Higginson, and L. H. Opie, *Circulation*, **67**, 24-30 (1983).
11. S. Penpargkul, A. Schwartz, and J. Scheuer, *Ibid.*, **54**, Suppl. 2, 114 (1976).
12. A. Quantanilha, *Biochem. Soc. Trans.*, **12**, № 3, 403 (1984).
13. L. D. Segel and D. T. Mason, *J. Appl. Physiol.*, **44**, 183-189 (1978).